Available online at www.sciencedirect.com
SCIENCE DIRECT®

Environment International 32 (2006) 412 – 416

ENVIRONMENT INTERNATIONAL

www.elsevier.com/locate/envint

Pesticide exposure and lung cancer mortality in Leningrad province in Russia

Won Jin Lee [a,b,*], Andrea Baccarelli [c], Maria Tretiakova [d], Sergey Gorbanev [e], Alexei Lomtev [e], Irina Klimkina [e], Vladimir Tchibissov [f], Olga Averkina [f], Mustafa Dosemeci [b]

[a] Department of Preventive Medicine, College of Medicine, Korea University, Seoul, Korea
[b] Division of Cancer Epidemiology and Genetics, National Cancer Institute, Rockville, MD, USA
[c] Department of Occupational and Environmental Health, University of Milan, Milan, Italy
[d] Department of Pathology, University of Chicago, Chicago, IL, USA
[e] Regional Center of Hygiene and Sanitation, St. Petersburg, Russia
[f] Leningrad Oblast Pathological Bureau, St. Petersburg, Russia

Received 1 August 2005; accepted 6 October 2005
Available online 8 November 2005

Abstract

This study was carried out to examine the association between pesticide exposure and lung cancer mortality. We conducted an autopsy based case-control study in Leningrad Province in Russia. A total of 540 lung cancer cases and 582 controls were identified among subjects who had died in the hospitals of the Leningrad province between 1993 and 1998. Using work history records, we assessed exposure to pesticide at the level of industry and job title. Unconditional logistic regression was used to calculate adjusted odds ratio for pesticide exposure and lung cancer mortality. There was no association between ever exposure to pesticide and lung cancer mortality overall (odds ratio = 1.06, 95% confidence interval = 0.82 – 1.36) and in both men (odds ratio = 1.11, 95% confidence interval = 0.84 – 1.46) and women (odds ratio = 0.74, 95% confidence interval = 0.37 – 1.46). We observed no statistically significant odds ratio by duration of pesticide exposure, intensity of pesticide exposure, and cumulative pesticide exposures with lung cancer mortality in both smokers and nonsmokers. Odds ratio also did not differ when the analysis was restricted to individuals who had exposure data with high confidence scores. Our findings suggest no associations between pesticide exposures and mortality of lung cancer in the population of the Leningrad province in Russia that deserves further evaluation.

Keywords: Lung neoplasms; Occupation; Pesticide exposure; Russia

1. Introduction

Lung cancer is the most common cause of cancer mortality in Russia and worldwide (Ferlay et al., 2001). While cigarette smoking is the most important cause for lung cancer, occupational exposures have been reported as risk factors for lung cancer (Blot and Fraumeni, 1996).

Increased risk for lung cancer among pesticide-exposed populations has been observed in several studies. In mortality and case-control studies of licensed pesticide applicators in the United States (Blair et al., 1983; Pesatori et al., 1994), the risk of lung cancer rose with the number of years licensed. In a survey of agricultural applicators in Germany, Barthel (1981) observed almost a two-fold excess mortality from lung cancer. A relationship between exposure to pesticide and lung cancer mortality was also observed in a cohort of workers from four manufacturing plants in Germany (Becher et al., 1996) and in a pooled analysis from 12 countries (Kogevinas et al., 1997). Similarly, the workers in the construction industry exposed to DDT had an excess risk of lung cancer after controlling for smoking in Uruguay (De Stefani et al., 1996) while orchardists exposed to DDT did not show increased lung cancer risk in a case-control study in the United States (Wicklund et al., 1988). Other several studies of pesticide applicators (Wang and MacMahon, 1979; MacMahon et al., 1988; Torchio et al., 1994; Fleming et al., 1999),

Abbreviations: CI, confidence interval; OR, odds ratio.

* Corresponding author. Department of Preventive Medicine, College of Medicine, Korea University, 126-1, 5-ka, Anam-Dong, Seongbuk-Gu, Seoul 136-705, Korea. Tel.: +82 2 920 6413; fax: +82 2 927 7220.
E-mail address: leewj@korea.ac.kr (W.J. Lee).

0160-4120/$ - see front matter © 2005 Elsevier Ltd. All rights reserved.
doi:10.1016/j.envint.2005.10.001

pesticide manufacturers (Coggon et al., 1986; Bond et al., 1988), and farmers (Forastiere et al., 1993; Settimi et al., 2001; McDuffie et al., 1990; Swaen et al., 2004), however, did not show any excess risk of lung cancer.

Inconsistencies between epidemiological studies may result from differences in study design, variation in pesticide exposure between different populations, or other underlying characteristics of the population, including both environmental factors and inherited susceptibility. The limited assessment of pesticide exposure, smaller sample size, and inadequate control of cigarette smoking in many of previous studies, underscores the need for improved investigations.

An autopsy-based case-control study was carried out in the Leningrad province in Russia to evaluate possible associations between lung cancer and occupational exposures. Here, we present results on pesticide exposure and lung cancer mortality.

2. Methods

2.1. Study subjects

In Leningrad province in Russia, an industrialized area with a population of 1.6 million subjects, post-mortem examinations were performed on approximately 95% of the subjects who died in the 88 local state hospitals. We identified 540 pathologically diagnosed lung cancer cases (474 men and 66 women), and 582 controls (453 men and 129 women) from the 1993–1998 autopsy records of the St. Petersburg Central Pathology Laboratory, which collects reports on all autopsies performed in the province. Controls were frequency matched to the cases by age (5-year group), gender (1:1 for men and 1:2 for women), geographical area (17 regions), and year of death and were selected among deceased subjects with autopsy-based diagnosis of non-cancer and non-smoking related diseases. The majority of causes of death of the control subjects were heart failure, diseases of the nervous system and digestive system. Gender-specific proportions of smokers among control subjects were comparable to the reported prevalence of tobacco use in the Russian Federation (Shafey et al., 2003). The study protocol was reviewed and approved by the Institutional Review Board of the United States National Cancer Institute.

2.2. Pesticide exposure assessment

Each individual was issued a personal record book, commonly called “Green Book” upon first employment in Russia. The workplace administration records in it detailed information, coded using the standard Russian occupational classification system, which includes the date of start and end of each job, industry and job title, factory name and department, as well as general data, such as marital status, education level, and total number of jobs. When a worker changes jobs, the Green Book is transferred to the new employer and updated. We retrieved the Green Book of all the study subjects either from family members of the deceased individuals, or from the archives of the hospital where the subjects had died. We also obtained health-related data, including information on smoking history, alcohol consumption and physical activity, from local health services and hygiene centers that routinely use standardized protocols to record them.

We estimated level of potential pesticide exposure for each job title in each industry, using Green Books’ personal occupational records through the expert judgment of an industrial hygienist (M. Dosemeci) together with Russian occupational hygiene specialists who are familiar with each local workplace. In the classification process, we reviewed all the existing literature and local measurements collected by the Russian hygienists. Since we were not able to identify specific pesticides, we assumed that estimating exposure to specific chemicals on the basis of very limited information would have produced large misclassification. Therefore we decided to classify exposure in general pesticide categories.

The level of pesticide exposure referred to the relative score among the exposed workers and was categorized as 0 (no exposure), 1 (low), 2 (intermediate), and 3 (high) based on the proximity of workers with pesticide exposures using the information on work activities and job descriptions. Agricultural and forestry workers were primarily considered as high pesticide exposed workers. The job title, industry, and their pesticide exposure level we categorized in this study are presented in Table 1. For each exposure, a confidence score reflecting the degree of certainty in the information retrieved (from 1=low to 3=high) was assigned. We constructed three pesticide exposure variables, all categorized into three for the statistical analysis by using each median value: (i) duration of pesticide exposure job (no exposure, $\leq$15 years, >15 years), (ii) pesticide intensity level by multiplying pesticide exposure level of industry by pesticide exposure level of job (no exposure, $\leq$4, >4), (iii) cumulative pesticide exposure as lifetime exposure by multiplying duration of pesticide exposure job by pesticide intensity level (no exposure, $\leq$70, >70).

2.3. Statistical analysis

We used unconditional logistic regression to calculate odds ratios (ORs) and 95% confidence intervals (CIs) with Stata software (version 8.0) (StataCorp., 2003). All significance tests were two-sided. The ORs for pesticide exposure were calculated using non-exposed subjects as a reference group.

All ORs were adjusted for age ($\leq$59, 60–69, $\geq$70), gender, and smoking (never, <1 pack/day, $\geq$1 pack/day). To control more fully for potential confounders, we also included variables for physical activity, education level, marital status, alcohol consumption, other agricultural agents such as animal dust, vegetable dust, silicon, gasoline, diesel, mineral fertilizer. Variables were retained in models as confounders when inclusion changed the value of the OR by more than 10% in any exposure category. In the final models, however, we retained a shorter list of potential confounders. We assigned scores to the categorical variables and treated them as continuous variables in logistic analysis to obtain tests for trend. We also calculated ORs including only subjects with high confidence score of pesticide exposure.

3. Results

The characteristics of the cases and controls are shown in Table 2. Compared with controls, lung cancer cases were significantly more men (87.8% versus 77.8%) as a result of the frequency matching by gender across all case groups, smokers (88.6% versus 67.1%), and alcohol drinkers (62.0% versus 48.2%). Education level, physical activity and marital status were similar between cases and controls.

Table 3 shows the adjusted ORs for lung cancer by pesticide exposure. There was no association between ever exposure to pesticide and lung cancer mortality overall (OR=1.06, 95% CI=0.82–1.36) and in both men (OR=1.11, 95% CI=0.84–1.46) and women (OR=0.74, 95% CI=0.37–1.46). We observed no statistically significant ORs by duration of pesticide exposure, intensity of pesticide exposure, and cumulative pesticide exposure with lung cancer mortality. In all analyses, we also obtained similar results among those who have exposure data of high confidence scores (data not shown).

We calculated ORs for lung cancer mortality with pesticide exposure stratified by smoking history to investigate potential modifying effects of smoking. We observed no significant risks of lung cancer in subjects exposed to pesticide among never, light, and heavy smokers (Table 4).

4. Discussion

We found no evidence of an increased risk of lung cancer mortality among population in the Leningrad province in Russia with the exposure of pesticide. The risk estimates were not modified by smoking history and also did not differ when

Table 1
Job title/industry and estimated level of pesticide exposure in the study subjects[a]

Industry/Job title	Pesticide exposure level	Cases	Controls
Agriculture	3	168	169
Forestry	3	11	14
Cattle-farm worker	3	16	20
Field-crop grower	3	7	17
Forest ranger	3	5	2
Breeder (swine, cattle)	3	5	1
Agronomist	3	2	3
Worker engaged in planting trees	3	1	1
Seed-grower	3	1	0
Forester	3	1	0
Plant growing worker	3	1	0
Chief agronomist	3	0	1
Gardener	3	0	1
Veterinarian assistant	3	0	1
Railroad transport	2	49	44
Defense industry	2	29	35
Lumber industry	2	30	24
Wood-processing industry	2	41	31
Tractorist	2	54	41
Odd-job worker	2	49	36
Loader-packer	2	43	24
Carpenter	2	36	34
Packer	2	6	7
Wood cutter	2	6	4
Uploader	2	5	1
Railroad repairman	2	4	2
Combiner	2	3	3
Railroad worker	2	3	3
Zoo technician	2	1	1
Timber-slide worker	2	1	1
Fuller	2	1	0
Veterinarian	2	0	2
Breeder (others)	2	0	1
Trade and public nutrition	1	55	51
Housing and communal services	1	62	68
General construction	1	73	83
Pulp and paper industry	1	8	10
Light industry	1	19	19
Food industry	1	35	24
Automobile driver	1	80	80
Metalworker	1	61	49
Supervisor	1	30	30
Warehouse man	1	7	15
Brigade-foreman	1	5	14
Inspector	1	4	5
Workshop chief deputy	1	3	2
Chairman	1	3	4
Railroad switchman	1	2	4
Safety measures engineer	1	2	1
Chief deputy	1	1	1
Deputy director	1	1	1
Train worker	1	1	0
Senior foreman	1	1	0
Quarantine inspector	1	1	0
Cellulose maker	1	0	1
Manager	1	0	1
Worker for mechanical cleaning	1	0	1
Hot-house worker	1	0	1

[a]The number of cases and controls is based on total jobs of individual, therefore, total numbers are more than subjects numbers.

the analysis was restricted to the subjects with high confidence score for pesticide exposure.

Our findings were consistent with other previous studies from pesticide applicators (Wang and MacMahon, 1979; MacMahon et al., 1988; Torchio et al., 1994; Fleming et al., 1999), pesticide manufacturers (Coggon et al., 1986; Bond et al., 1988), and farmers (Forastiere et al., 1993; Settimi et al., 2001; McDuffie et al., 1990; Swaen et al., 2004). However, the plausibility of a link between pesticide exposure and lung cancer has been suggested by previous studies. The International Agency for Research on Cancer classified 26 pesticides as having sufficient evidence of carcinogenicity and 19 pesticides as having limited evidence in animals (International Agency for Research on Cancer, 1991). Bolognesi reviewed literatures and concluded that positive association between occupational pesticide exposure and the presence of chromosome aberrations, sister-chromatid exchange and micronuclei has been detected in the majority of the studies (Bolognesi, 2003). Recently, the Agricultural Health Study, a large cohort study of pesticide applicators in Iowa and North Carolina, reported some pesticides widely used currently in the United States have been significantly associated with lung cancer risk (Alavanja et al., 2004; Lee et al., 2004).

The different results in each study may from possible misclassification of pesticide exposure. In our study, misclas-

Table 2
Characteristics of demographic, life style, educational and marital status of cases and control

Characteristics	Cases (N=540)		Controls (N=582)	
	Number[a]	%	Number[a]	%
Age				
≤59	177	32.8	175	30.1
60–69	255	47.2	276	47.4
≥70	108	20.0	131	22.5
Gender				
Male	474	87.8	453	77.8
Female	66	12.2	129	22.2
Smoking				
Never	61	11.4	190	32.9
Light (<1 pack/day)	208	38.7	217	37.5
Heavy (≥1 pack/day)	268	49.9	171	29.6
Alcohol drinking				
No/little	205	38.0	300	51.8
Every week	190	35.3	170	29.4
Every day	144	26.7	109	18.8
Education				
<High school	104	19.4	98	17.1
High school	321	60.0	335	58.4
>High school	110	20.6	141	24.5
Marital status				
Single	35	6.5	40	6.9
Married	385	71.8	408	70.2
Divorced/widow	116	21.7	133	22.9
Physical activity				
Regular exercise	11	2.2	14	2.5
Some exercise, irregular	60	11.8	50	8.9
Gardening	218	42.7	251	44.9
Little or no activity	221	43.3	244	43.7

[a] Missing data for some questions are responsible for difference in total cell counts.

Table 3
Odds ratios (ORs) and 95% confidence intervals (95% CI) for lung cancer mortality by pesticide exposure

Pesticide exposure	All subjects				Men				Women			
	Ca.	Co.	OR[a]	95% CI	Ca.	Co.	OR[a]	95% CI	Ca.	Co.	OR[a]	95% CI
No exposure	306	359	1.0	Ref[b]	256	270	1.0	Ref[b]	50	89	1.0	Ref[b]
Ever exposure	234	223	1.06	0.82–1.36	218	183	1.11	0.84–1.46	16	40	0.74	0.37–1.46
Duration (years)[c]												
Low (≤15)	127	115	1.10	0.81–1.50	118	91	1.20	0.86–1.69	9	24	0.67	0.28–1.60
High (>15)	107	108	1.00	0.73–1.40	100	92	1.01	0.71–1.44	7	16	0.83	0.31–2.20
Intensity[d]												
Low (≤4)	117	111	1.07	0.78–1.47	109	94	1.10	0.78–1.55	8	17	0.87	0.34–2.20
High (>4)	117	112	1.04	0.76–1.43	109	89	1.12	0.79–1.58	8	23	0.64	0.26–1.56
Cumulative score[e]												
Low (≤70)	133	117	1.15	0.85–1.56	123	97	1.19	0.85–1.66	10	20	0.96	0.41–2.28
High (>70)	101	106	0.96	0.69–1.33	95	86	1.02	0.71–1.45	6	20	0.53	0.20–1.42

[a] ORs were adjusted for age, gender, and smoking status.
[b] Reference category.
[c] Pesticide duration (years of pesticide exposure job).
[d] Pesticide intensity (pesticide exposure level of industry × pesticide exposure level of job).
[e] Cumulative pesticide score (years of pesticide exposure job × pesticide intensity level).

sification of exposure may also have occurred because work histories were available only at the industry or job title level, although the assessment of pesticide was based on a detailed work history. If the exposure among subjects in an industry or job title is not homogeneous, then unexposed subjects are classified as potentially exposed (and vice versa). We also could not incorporate the changes in exposure over time. These potential misclassifications would be resulting in a tendency to underestimate the risk, if there is one. We addressed the potential misclassification of exposure by restriction of the study population to those with high confidence and found similar results with those of total subjects.

In our study, it was not possible to investigate the risk of lung cancer with exposure to specific pesticides. This is a potential source of misclassification because not all pesticides have carcinogenic properties. The effect of pesticide exposure may be diluted if non-carcinogenic pesticides are considered together with other carcinogenic pesticides.

Although smoking data in our study had been recorded before the study and were, thus, not affected by recall bias, we cannot exclude that misclassification may have occurred. In particular, tobacco use may have been underreported by female subjects because smoking was not considered proper for women in Russia.

Although we selected controls randomly from subjects who had died non-cancer and non-smoking related diseases, choosing deceased controls may misrepresent the exposure distribution in the source population if the exposure causes or prevent death in a substantial number of people or if it is associated with another factor that does (Rothman and Sandler, 1998).

Another possible limitation of the study is the lack of information on histological type of lung cancer. This may limit our ability to capture histology-specific lung cancer risk by pesticide exposure. We are in process to obtain histological information on lung cancer to overcome this limitation.

Table 4
Odds ratios (ORs) and 95% confidence intervals (95% CI) for lung cancer mortality by pesticide exposure and smoking history

Pesticide exposure	Never smokers				Light smokers (<1 pack/day)				Heavy smokers (≥1 pack/day)			
	Ca.	Co.	OR[a]	95% CI	Ca.	Co.	OR[a]	95% CI	Ca.	Co.	OR[a]	95% CI
No exposure	43	133	1.0	Ref[b]	123	130	1.0	Ref[b]	140	92	1.0	Ref[b]
Ever exposure	18	57	1.06	0.55–2.04	85	87	1.06	0.71–1.58	128	79	1.07	0.72–1.57
Duration (years)[c]												
Low (≤15)	10	24	1.38	0.59–3.23	47	58	0.90	0.57–1.44	68	33	1.40	0.85–2.30
High (>15)	8	33	0.82	0.34–1.97	38	29	1.36	0.79–2.36	60	46	0.83	0.52–1.33
Intensity[d]												
Low (≤4)	7	28	0.83	0.33–2.11	45	49	0.98	0.60–1.59	64	34	1.24	0.75–2.03
High (>4)	11	29	1.28	0.57–2.90	40	38	1.17	0.70–1.97	64	45	0.94	0.59–1.49
Cumulative score[e]												
Low (≤70)	9	28	1.11	0.47–2.62	51	54	1.03	0.65–1.64	71	35	1.36	0.83–2.20
High (>70)	9	29	1.01	0.43–2.40	34	33	1.11	0.64–1.93	57	44	0.84	0.52–1.35

[a] ORs were adjusted for age and gender.
[b] Reference category.
[c] Pesticide duration (years of pesticide exposure job).
[d] Pesticide intensity (pesticide exposure level of industry × pesticide exposure level of job).
[e] Cumulative pesticide score (years of pesticide exposure job × pesticide intensity level).

Despite the limitations above, our study is unique in that it was based on autopsy lung cancer cases and controls who had died in the hospitals of the Leningrad region covering more than 95% of hospital cases. Because the subjects' selection was conducted using records from the St. Petersburg Central Pathology Laboratory archives, all cases had reliable lung cancer diagnoses. The availability of autopsy data represents a rare opportunity to obtain highly accurate causes of death and reduce disease misclassification in epidemiologic analysis. We could also use Green Book, a unique personal occupational record in Russia, for the assessment of pesticide lifetime exposure.

In summary, we found no significant association between pesticide exposure and mortality of lung cancer among populations in the Leningrad province in Russia. Considering the widespread use of pesticide and high incidence of lung cancer, however, further studies, particularly with carefully defined individual pesticides use and genetic information on lung cancer susceptability, are needed.

References

Alavanja MC, Dosemeci M, Samanic C, Lubin JH, Lynch CF, Knott C, et al. Pesticides and lung cancer risks in the Agricultural Health Study Cohort. Am J Epidemiol 2004;160:876–85.

Barthel E. Increased risk of lung cancer in pesticide-exposed male agricultural workers. J Toxicol Environ Health 1981;8:1027–40.

Becher H, Flesch-Janys D, Kauppinen T, Kogevinas M, Steindorf K, Manz A, et al. Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins. Cancer Causes Control 1996;7:312–21.

Blair A, Grauman DJ, Lubin JH, Fraumeni Jr JF. Lung cancer and other causes of death among licensed pesticide applicators. J Natl Cancer Inst 1983;71:31–7.

Blot W, Fraumeni Jr JF. Cancers of the lung and pleura. In: Schottenfeld D, Fraumeni Jr JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 637–65.

Bolognesi C. Genotoxicity of pesticides: a review of human biomonitoring studies. Mutat Res 2003;543:251–72.

Bond GG, Wetterstroem NH, Roush GJ, McLaren EA, Lipps TE, Cook RR. Cause specific mortality among employees engaged in the manufacture, formulation, or packaging of 2,4-dichlorophenoxyacetic acid and related salts. Br J Ind Med 1988;45:98–105.

Coggon D, Pannett B, Winter PD, Acheson ED, Bonsall J. Mortality of workers exposed to 2 methyl-4 chlorophenoxyacetic acid. Scand J Work Environ Health 1986;12:448–54.

De Stefani E, Kogevinas M, Boffetta P, Ronco A, Mendilaharsu M. Occupation and the risk of lung cancer in Uruguay. Scand J Work Environ Health 1996;22:346–52.

Ferlay J, Bray F, Pisani P, Parkin DM. Cancer incidence, mortality and prevalence worldwide. GLOBOCAN 2000. Lyon, France: International Agency for Research on Cancer; 2001.

Fleming LE, Bean JA, Rudolph M, Hamilton K. Mortality in a cohort of licensed pesticide applicators in Florida. Occup Environ Med 1999; 56:14–21.

Forastiere F, Quercia A, Miceli M, Settimi L, Terenzoni B, Rapiti E, et al. Cancer among farmers in central Italy. Scand J Work Environ Health 1993;19:382–9.

International Agency for Research on Cancer. Occupational exposures in insecticide application, and some pesticide. IARC Monogr Eval Carcinog Risks Hum 1991;53:5–586.

Kogevinas M, Becher H, Benn T, Bertazzi PA, Boffetta P, Bueno-de-Mesquita HB, et al. Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins An expanded and updated international cohort study. Am J Epidemiol 1997;145:1061–75.

Lee WJ, Blair A, Hoppin JA, Lubin JH, Rusiecki JA, Sandler DP, et al. Cancer incidence among pesticide applicators exposed to chlorpyrifos in the Agricultural Health Study. J Natl Cancer Inst 2004;96:1781–9.

MacMahon B, Monson RR, Wang HH, Zheng TZ. A second follow-up of mortality in a cohort of pesticide applicators. J Occup Med 1988;30: 429–32.

McDuffie HH, Klaassen DJ, Dosman JA. Is pesticide use related to the risk of primary lung cancer in Saskatchewan? J Occup Med 1990;32:996–1002.

Pesatori AC, Sontag JM, Lubin JH, Consonni D, Blair A. Cohort mortality and nested case-control study of lung cancer among structural pest control workers in Florida (United States). Cancer Causes Control 1994; 5:310–8.

Rothman KJ, Sandler G, 1998. Case-control studies. 2nd ed. Philadelphia: Lippincott-Raven Publishers; 1998. p. 103.

Settimi L, Comba P, Bosia S, Ciapini C, Desideri E, Fedi A, et al. Cancer risk among male farmers: a multi-site case-control study. Int J Occup Med Environ Health 2001;14:339–47.

Shafey O, Dolwick S, Guindon GE. Russian Federation. Tobacco control country profiles 2003. Atlanta, GA: American Cancer Society; 2003. p. 330–1.

StataCorp. Stata reference manual release, vol. 8. Texas: Stata Press; 2003.

Swaen GM, Van Amelsvoort LG, Slangen JJ, Mohren DC. Cancer mortality in a cohort of licensed herbicide applicators. Int Arch Occup Environ Health 2004;77:293–5.

Torchio P, Lepore AR, Corrao G, Comba P, Settimi L, Belli S, et al. Mortality study on a cohort of Italian licensed pesticide users. Sci Total Environ 1994;149:183–91.

Wang HH, MacMahon B. Mortality of pesticide applicators. J Occup Med 1979;21:741–4.

Wicklund KG, Daling JR, Allard J, Weiss NS. Respiratory cancer among orchardists in Washington State, 1968 to 1980. J Occup Med 1988; 30:561–4.